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ADISINSIGHT REPORT

Catumaxomab: First Approval

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Abstract

Catumaxomab (Korjuny®) is a first-in-class bispecific trifunctional rat-mouse hybrid monoclonal antibody currently under development with Lindis Biotech for malignant ascites, and bladder, gastric and ovarian cancers. It binds epithelial cell adhesion molecule (EpCAM) on tumour cells and CD3 on T cells, while its Fc domain engages Fcγ receptor-positive accessory cells, bringing immune and tumour cells into close proximity to enhance tumour cell killing through multiple immunological mechanisms. Initially approved in the EU on 20 April 2009 for malignant ascites in adults with EpCAM+ carcinomas when standard therapy was unavailable or no longer feasible, catumaxomab was marketed by Fresenius Biotech GmbH (later Neovii Biotech GmbH) before being withdrawn on 2 June 2017 for commercial reasons. Lindis Biotech later acquired the rights and pursued reapproval. On 11 February 2025, catumaxomab was approved in the EU for the intraperitoneal treatment of malignant ascites in adults with EpCAM+ carcinomas who are not eligible for further systemic anticancer therapy. This article summarizes the milestones in the development of catumaxomab leading to this new approval.

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Catumaxomab (Korjuny®): Key Points

A first-in-class trifunctional monoclonal antibody being developed by Lindis Biotech for malignant ascites and bladder, gastric, and ovarian cancers.

Received reapproval on 11 February 2025 in the EU.

Approved for the intraperitoneal treatment of malignant ascites in adults with EpCAM+ carcinomas who are not eligible for further systemic anticancer therapy.

1 Introduction

Catumaxomab (Korjuny®) is a first-in-class bispecific trifunctional rat-mouse hybrid monoclonal antibody with distinct antigen-binding sites for epithelial cell adhesion molecule (EpCAM) and CD3, along with a functional Fc domain [1–4]. It is currently being developed by Lindis Biotech for the treatment malignant ascites, as well as bladder, gastric and ovarian cancers. Malignant ascites, a severe complication of advanced-stage cancers, leads to significant symptomatic burden, discomfort and impaired quality of life [5]. Until recently, no approved drug therapies were available for its treatment. EpCAM is expressed in tumour cells in malignant effusions [6], making it an attractive target for antibody therapy.

Catumaxomab received approval on 11 February 2025 in the EU for the intraperitoneal treatment of malignant ascites in adults with EpCAM+ carcinomas, who are not eligible for further systemic anticancer therapy [3, 7]. Treatment consists of four intraperitoneal infusions of 10 µg, 20 µg, 50 µg and 150 µg on days 0, 3, 7 and 10, respectively, under the supervision of a physician experienced in anticancer drugs [3]. The recommended infusion time is 6 h for the first infusion and a minimum of 3 h for subsequent infusions. The interval between infusions may be extended if necessary to reduce adverse reactions, but the total treatment period must not exceed 21 days. Patients require close medical supervision for at least 24 h after the first infusion. For subsequent

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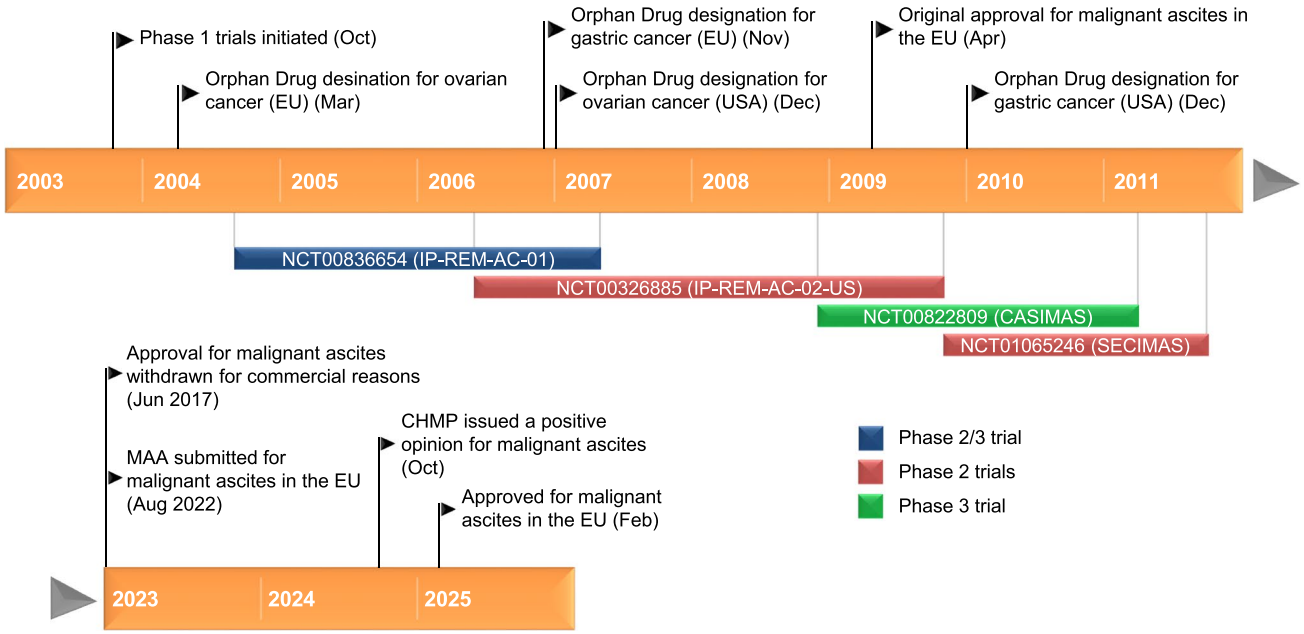

Key milestones in the development of catumaxomab. *CHMP* Committee for Medicinal Products for Human Use, *MAA* Marketing Authorisation Application

doses, hospitalisation for at least 6 h is recommended, with extended monitoring at the physician's discretion [3].

Catumaxomab was originated by Trion Pharma GmbH using its Triomab® platform. Fresenius Biotech GmbH (now Neovii Biotech GmbH) developed the antibody and received marketing authorisation in the EU under the brand name Removab® on 20 April 2009 for intraperitoneal treatment of malignant ascites in adults with EpCAM+ carcinomas, where standard therapy is unavailable or no longer feasible [8]. The product has not been marketed since 2014 and was withdrawn from the EU on 2 June 2017 for commercial reasons [7]. Lindis Biotech GmbH has since acquired the rights to catumaxomab and pursued a new marketing authorisation application.

Catumaxomab is being evaluated in clinical trials for non-muscle-invasive bladder [9, 10], gastric [11, 12] and ovarian [13, 14] cancers. It has Orphan Drug Designation in the EU [15, 16] and the USA [17] for the treatment of ovarian and gastric cancers.

1.1 Company Agreements

In November 2024, Lindis Biotech entered into a licensing agreement with Pharmanovia for exclusive rights to market and launch catumaxomab for treating malignant ascites in Europe [18]. In 2017, LintonPharm in-licensed four Triomab® antibodies from Lindis Biotech for development and commercialization in the Asia-Pacific region [19].

In 1998, Trion Pharma GmbH and Fresenius Biotech GmbH signed a collaboration and licensing agreement for tri-functional bispecific monoclonal antibodies. Fresenius gained global rights to market, distribute and develop catumaxomab. In January 2011, Swedish Orphan Biovitrum and Fresenius Biotech signed a distribution agreement for catumaxomab in 15 European countries over 7 years [20]. In June 2013, Fresenius Biotech was sold to Neopharm and renamed Neovii Biotech GmbH, which now operates as a Neopharm subsidiary [21].

2 Scientific Summary

2.1 Pharmacodynamics

Catumaxomab binds EpCAM on epithelial tumour cells and CD3 on T cells, while its Fc domain activates Fcγ receptor-positive accessory cells, bringing tumour cells, T cells and accessory immune cells into close proximity [1–4]. This leads to improved tumour cell elimination through different mechanisms such as T-cell activation, granzyme/perforin-mediated killing, antibody-dependent cell-mediated cytotoxicity, complement-dependent cytotoxicity and phagocytosis [1–4].

The pharmacodynamic effects of catumaxomab have been reviewed elsewhere [22]. Briefly, it demonstrated antitumour activity in vitro and in vivo, effectively killing EpCAM+ tumour cells regardless of primary tumour

type [3]. In an immunocompromised mouse model of ovarian carcinoma, intraperitoneal catumaxomab with human peripheral blood mononuclear cells delayed tumour development [3].

In malignant ascites samples from patients participating in the pivotal phase 2/3 trial (Sect. 2.3), intraperitoneal catumaxomab treatment reduced EpCAM+ tumour cells, decreased the ratio of EpCAM+ tumour cells to CD45+ leukocytes, eliminated CD133+/EpCAM+ cancer stem cells, lowered tumour cell counts at the repuncture visit, reduced vascular endothelial growth factor levels and enhanced T-cell activation [23, 24]. In vitro, catumaxomab significantly reduced tumour cell numbers and induced a strong proinflammatory cytokine response, including increased levels of IFN-γ, TNF-α, IL-2, and IL-6 in ascites cells [23, 24]. Catumaxomab also activated natural killer cells and macrophages in malignant ascites in a separate study [25].

2.2 Pharmacokinetics

The pharmacokinetics of intraperitoneal catumaxomab were assessed in an open-label, multicentre phase 2 trial (IP-REM-PK-01-EU) in 13 patients with symptomatic malignant ascites due to EpCAM+ carcinomas [3, 26]. Patients received four 6-h constant-rate intraperitoneal infusions at doses of 10, 20, 50 and 150 µg. Catumaxomab was detectable in both ascitic fluid and plasma, with concentrations increasing with dose and number of infusions. However, inter-individual variability was high due to differences in ascites volume and malignant cell burden. Following intraperitoneal infusion, catumaxomab was immediately available in ascitic fluid, with mean and median C_{max} values of 7122 and 3270 pg/mL, respectively. After binding to target cells in the peritoneal cavity, residual catumaxomab reaches systemic circulation in intact form. Plasma concentrations were much lower (mean C_{max} 0.5 ng/mL) and peaked after the fourth infusion. Systemic catumaxomab undergoes proteolytic catabolism, with a mean plasma half-life of 2.5 days [3, 26].

2.3 Therapeutic Trials

In a phase 1/2 trial (STP-REM-01), intraperitoneal catumaxomab administered as 6-h infusions of 5–200 µg over 9 to 13 days significantly reduced ascites flow rate in 23 patients with malignant ascites due to advanced ovarian cancer [27]. Of these, 22 patients did not require paracentesis between the final infusion and day 37. Based on the maximum tolerated dose identified in this study, a 4-dose series of 10-20-50-150 µg was recommended for further clinical trials [27].

In a single-arm phase 2 trial (IP-REM-AC-02-US), catumaxomab demonstrated preliminary efficacy in patients with malignant ascites due to chemotherapy-refractory ovarian cancer [28]. Thirty-two patients received four 3-h intraperitoneal catumaxomab infusions (10, 20, 50 and 150 µg within 10 days). Seven patients (23%) achieved the primary endpoint of at least a 4-fold increase in puncture-free interval relative to the pre-treatment interval. The median puncture-free interval increased from 12 to 27.5 days, while the median time to first therapeutic puncture increased 4-fold (12 to 52 days). Median puncture-free survival (PuFS) and overall survival (OS) were 29.5 and 111 days, respectively. Catumaxomab also improved ascites symptoms [28].

Features and properties of catumaxomab

Alternative names	Anti-CD3 anti-EpCAM monoclonal antibody; anti-EpCAM anti-CD3 monoclonal antibody; KORJUNY; LP-000; Removab
Class	Antineoplastics; bispecific antibodies; immunoglobulin Fc fragments; immunotherapies
Mechanism of action	Antibody-dependent cell cytotoxicity; cytotoxic T lymphocyte stimulants
Route of administration	Intraperitoneal
Pharmacodynamics	Kills tumour cells through different mechanisms such as T-cell activation, granzyme/perforin-mediated killing, antibody-dependent cell-mediated cytotoxicity, complement-dependent cytotoxicity and antibody-dependent cellular phagocytosis
Pharmacokinetics	Detectable in ascitic fluid immediately after intraperitoneal infusion, with concentrations increasing with dose and number of infusions; low systemic exposure; mean plasma half-life 2.5 days
Most frequent adverse events	Pyrexia, nausea, vomiting, abdominal pain
ATC codes	
WHO ATC code	L01F-X03 (Catumaxomab)
EphMRA ATC code	L1G (Monoclonal Antibody Antineoplastics)
Chemical name	Immunoglobulin G2a, anti-(human antigen 17-1A) (mouse monoclonal Ho-3/TP-A-01/TPBs01 heavy chain), disulfide with mouse monoclonal Ho-3/TP-A-01/TPBs01 light chain, disulfide with immunoglobulin G2b anti-(human CD3 (antigen)) (rat monoclonal 26/II/6-1.2/TPBs01 heavy chain), disulfide with rat monoclonal 26/II/6-1.2/TPBs01 light chain

2.4 Pivotal Trial

Catumaxomab combined with paracentesis provided clinically significant benefits in treating malignant ascites from epithelial cancer in a randomized, open-label phase 2/3 trial (IP-REM-AC-01) [24]. Eligible patients were adults (≥ 18 years) with histologically confirmed epithelial cancer and EpCAM+ tumour cells in ascites fluid, requiring symptomatic paracentesis. A total of 258 patients were randomly assigned in a 2:1 ratio to catumaxomab plus paracentesis or paracentesis alone, stratified by ovarian ($n = 85, 44$) and nonovarian ($n = 85, 44$) cancer. Catumaxomab was administered as four intraperitoneal infusions at doses of 10, 20, 50 and 150 µg on days 0, 3, 7, and 10, respectively. Both groups received one baseline paracentesis, with repuncture performed as needed. The primary efficacy endpoint was PuFS, defined as the composite of time to the first therapeutic puncture or death after treatment, whichever occurred first, assessed in the intent-to-treat population. Following the primary endpoint, patients were monitored for OS. Baseline demographic and clinical characteristics were comparable between treatment groups within each stratum. The most common cancer types in the nonovarian stratum were gastric (51%), breast (10%), pancreas (7%), colon (6%) and endometrial (5%) cancers. The PuFS analysis included 133 (66%) punctures and 68 (34%) deaths [24].

Median PuFS was significantly ($p < 0.0001$) longer in the catumaxomab plus paracentesis group compared with the paracentesis-only group in the overall population (46 vs 11 days; hazard ratio [HR] 0.2254; 95% CI 0.185–0.350), ovarian stratum (52 vs 11 days) and nonovarian stratum (37 vs 14 days) [24]. A consistent PuFS benefit was observed in patients with and without distant metastases [24]. The primary endpoint results are supported by the secondary endpoints [24]. The median time to the next therapeutic paracentesis was significantly ($p < 0.0001$) longer in the catumaxomab plus paracentesis group compared with the paracentesis-only group in the overall population (77 vs 13 days; HR 0.169; 95% CI 0.114–0.251), ovarian stratum (71

Key clinical trials of catumaxomab

Drug(s)	Indication	Phase	Status	Location(s)	Identifier	Sponsor
Catumaxomab, paracentesis	Malignant ascites due to EpCAM+ cancer	2/3	Completed	Europe	IP-REM-AC-01; EudraCT 2004-000723-15; NCT00836654	Fresenius Biotech
Catumaxomab	Malignant ascites due to ovarian cancer	1/2	Completed	Europe	STP-REM-01	Trion Pharma
Catumaxomab, prednisolone	Malignant ascites due to epithelial cancer	3b	Completed	Europe	IP-CAT-AC-03; CASIMAS; NCT00822809	Fresenius Biotech
Catumaxomab	Malignant ascites due to epithelial cancer	2	Completed	Germany	IP-CAT-AC-04; SECIMAS EudraCT 2009-014076-22; NCT01065246	Fresenius Biotech
Catumaxomab	Malignant ascites due to ovarian cancer	2	Completed	USA	IP-REM-AC-02-US NCT00326885	Fresenius Biotech
Catumaxomab	Malignant ascites	2	Completed	Europe	IP-REM-PK-01-EU; EudraCT 2005-001700-39	Fresenius Biotech
Catumaxomab	Non-muscle-invasive bladder cancer	1/2	Unknown	China	LP0190512; NCT04799847	LintonPharm
Catumaxomab	Non-muscle-invasive bladder-cancer	1	Completed	Germany	CATUNIBLA; EudraCT 2019-002850-22 NCT04819399	Lindis Biotech
Catumaxomab	Gastric cancer	2	Completed	Europe	IP-CAT-GC-03; EudraCT 2006-002727-16 NCT00464893	Fresenius Biotech
Catumaxomab	Gastric cancer	2	Completed	Germany	IP-REM-GC-02; NCT00352833	Fresenius Biotech
Catumaxomab, Investigator's choice of treatment	Gastric peritoneal carcinomatosis	1b, 3	1b completed	Asia	LP0190415; NCT04222114	LintonPharm
Catumaxomab	Ovarian cancer	2	Completed	USA	IP-CAT-OC-01; NCT00377429	Fresenius Biotech
Catumaxomab	Ovarian cancer	2	Completed	Austria, Germany	IP-CAT-OC-02 NCT00563836	Fresenius Biotech

vs 11 days; HR 0.152; 95% CI 0.088–0.260) and nonovarian stratum (80 vs 15 days; HR 0.183; 95% CI 0.101–0.331). The gastric cancer subgroup showed the greatest benefit (118 vs 15 days; HR 0.143, 95% CI 0.057–0.359). The median OS did not differ significantly between the groups in the overall population (72 vs 68 days; HR 0.723; 95% CI 0.498–1.048); however, a significant OS benefit with catumaxomab was observed in the gastric cancer subgroup (71 vs 44 days; $p = 0.0313$). At 8 days after treatment, fewer patients in the catumaxomab plus paracentesis group exhibited signs and symptoms of ascites compared with the paracentesis-only group [24].

A post hoc analysis indicated that patients who developed human antimouse antibodies (HAMAs) 8 days after the fourth dose of catumaxomab experienced greater clinical benefits compared to those who did not [29]. Significant differences were observed between HAMAs-positive and -negative patients for median PuFS (64 vs 27 days; HR 0.330; $p < 0.0001$), median time to next puncture (104 vs 46 days; HR 0.307; $p = 0.0002$) and median OS (129 vs 64 days; HR 0.433; $p = 0.0003$) [29]. Another post hoc analysis identified baseline relative lymphocyte count (RLC) as a potential independent prognostic biomarker for catumaxomab efficacy [30]. In patients with RLC > 13% at screening, catumaxomab plus paracentesis significantly prolonged median OS compared to paracentesis alone (109 vs 68 days; $p = 0.0072$). No significant between-group difference in OS was observed in patients with RLC ≤ 13%. Additionally, in the catumaxomab plus paracentesis group, median PuFS was significantly longer in those with RLC > 13% than those with RLC ≤ 13% (49 vs 31 days; $p = 0.0027$) [30].

Catumaxomab treatment delayed health-related quality of life (QoL) deterioration, as assessed by the EORTC QLQ-C30 in the phase 2/3 study [31]. Median time to deterioration was shorter in the paracentesis-only group (19–26 days) than in the catumaxomab plus paracentesis group (47–49 days). Significant ($p < 0.001$) differences between groups were seen for all QoL scores of primary interest: emotional functioning, global QoL, fatigue, nausea and vomiting, pain, dyspnoea, sleep disturbance and appetite loss [31].

2.5 Adverse Events

Catumaxomab had an acceptable tolerability and safety profile in patients with malignant ascites secondary to epithelial cancers in the phase 2/3 trial [24]. The adverse events (AEs) with catumaxomab were manageable and generally reversible. The longer AE observation period for catumaxomab plus paracentesis due to prolonged PuFS makes comparisons with paracentesis alone misleading. In the catumaxomab plus paracentesis group ($n = 157$), 85% of patients experienced catumaxomab-related AEs (mostly mild to moderate in severity), with 15% experiencing catumaxomab-related serious AEs. The most common catumaxomab-related AEs (any grade) were cytokine release-related symptoms (pyrexia 60.5%, nausea 33.1%, vomiting 27.4%) and abdominal pain (42.7%). Other catumaxomab-related AEs (any grade) included lymphopenia (14%), increased C-reactive protein (14.6%), chills (13.4%), elevated gamma-glutamyl transferase (11.5%), fatigue (10.8%), diarrhoea (10.2%), leucocytosis (10.2%), tachycardia (9.6%), anorexia (8.9%), increased blood alkaline phosphatase (8.9%), anaemia (8.9%), hypotension (8.3%), elevated aspartate aminotransferase (7.6%), alanine aminotransferase (6.4%), ileus (6.4%), and pain (5.1%). Laboratory abnormalities were rarely clinically relevant and were generally reversible. The AE profile of catumaxomab in the ovarian and nonovarian cancer patients was similar, with no distinctive pattern corresponding to specific infusions [24].

In an integrated safety analysis of 517 patients from 11 catumaxomab studies (including the phase 2/3 trial), the most commonly reported adverse reactions were pyrexia (62%), abdominal pain (42%), nausea (41%) and vomiting (38%) [3]. The most common serious adverse reactions were systemic inflammatory response syndrome and hepatic failure. Of the patients included in this analysis, 293 received catumaxomab intraperitoneally as a 6-h infusion and 224 received it as a 3-h infusion [3].

In a phase 3b trial (CASIMAS) in patients with malignant ascites due to epithelial cancer, prednisolone 25 mg given before four 3-h intraperitoneal catumaxomab infusions did not significantly reduce the intensity of the main AEs (pyrexia, nausea, vomiting and abdominal pain) or the frequency of catumaxomab-related AEs and grade ≥ 3 AEs [32]. A single-arm phase 1/2 trial (SECIMAS) found that a second cycle of catumaxomab was feasible in patients who had already received a first cycle of four infusions in the CASIMAS trial [33].

2.6 Companion Diagnostic

Before starting catumaxomab treatment, patients should be tested for EpCAM positivity (≥ 400 EpCAM+ cells per 10^6 analysed ascites cells) using a CE-marked in vitro diagnostic test with the appropriate intended purpose. If unavailable, a validated alternative test should be used.

2.7 Ongoing Clinical Trials

As of March 2025, there are no ongoing clinical trials of catumaxomab.

3 Current Status

On 11 February 2025, catumaxomab was approved in the EU for the intraperitoneal treatment of malignant ascites in adults with EpCAM+ carcinomas who are not eligible for further systemic anticancer therapy [3, 7].

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Declarations

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Ethics Approval, Consent to Participate, Consent to Publish, Availability of Data and Material, Code Availability Not applicable.

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